come under the category of and represent one phase of mental alienation. This so-called reasoning insanity is not properly an insanity, but is one of the necessary periods of mental trouble, whatever it may be or become. There also exists, according to the author, a stage of incubation in which the insanity is not yet characterized, and in which it commences with incomplete manifestations.

This reasoning vesanic disorder marks the evolution or involution of every classified insanity, and it may exist alone and disappear before the appearance of the ordinary attack.

Bigot divides this *folie raisonnante* into lucid, para-lucid, and pseudo-lucid forms.

Lucidity. The patient is more or less clearly aware of the morbid conception; if he conceals it we are unable to detect it, if he acknowledges it, it is in such a way that we hesitate to believe him insane.

Pseudo-lucidity. The patient is just conscious of his insanity. He accepts the insane notion, but understands that it is for his interest to hide it.

Para-lucidity. The patient does not conceal his delusion in which he maintains an obstinate faith, to spare himself from *ennui*.

Bigot also classifies those predisposed. He distinguishes intellectual and moral anomalies, of which he separates two types (the insufficient and the unstable) from that which he calls the direct forms, passional anomalies or manias, and the indirect or oblique mental disorders.

En resumé, says the author, there are reasoning maniacs, there is no reasoning insanity. The definite attack is frequently slow or brief, and this gives a great value to the intermediate states. Those patients who remain very long in an uncertain mental condition are most frequently abnormal forms of insanity. There are prolonged lucid intervals in every kind of mental alienation; there are good, bad, and indifferent. Between reason and confirmed insanity there is every shade of reasoning power; *natura non facit saltus*.

Dementia Paralytica.—W. Jessen (*Centralblatt*, 31st March, 1877) examined microscopically the pons and medulla in two cases of dementia paralytica, although no lesions were seen with the unaided eye. The morbid changes were considerable, especially in the region of the inferior decussation of the pyramids. They consisted in proliferation of the epithelium lining the central canal, and in destruction of the nerve-fibres and ganglionic cells.

Optic Neuritis in Acute Infantile Meningitis.—Dr. H. Parinaud, *Brochure*, Paris, 1877, (noticed in *Gaz. des Hopitaux*). *Conclusions.* Optic neuritis in the acute meningitis of infancy has all the clinical and anatomical characters of strangulated neuritis, such as we observe in the different conditions in which the intra-cranial pressure is increased. It is

not the result of any inflammatory alterations affecting the optic nerves in their intra-cranial course, but is due to the hydrocephalus, which is a frequent complication of acute meningitis, and which always accompanies the neuritis. The œdema of the optic nerve that characterizes the alteration, improperly designated neuritis, appears to us to be of the same nature as the cerebral œdema observed under the same conditions, and produced by an obstruction to the lymphatic circulation.

SENILE TREMOR.—At the session of the Soc. Méd. des Hopitaux, July 13, (rep. in *L'Union Médicale*), M. Luys offered a communication in regard to that special form of trembling which is usually described as peculiar to old age, and is known under the title of senile tremor. He maintained that this as a distinct form, and as peculiar to old age, did not really exist; in ten years careful observation of senile pathology in his service, both in the Bicetre and in the Salpetrière, and he had never observed any tremor in healthy old persons. He considered that senility was by no means necessarily accompanied with tremor. He had had, among others, under observation, a woman ninety-nine years of age who showed no signs of tremor. He attributed the partial tremors which existed in certain cases in the muscles of the hand and those of the neck, to incomplete forms of paralysis agitans, and to localized sclerosis in certain portions of the medulla or the pons.

THE HEAD SYMPTOMS IN LOCOMOTOR ATAXY.—The following is the analysis of a recent memoir by Dr. A. Pierret (52 pp. Paris, J. B. Bailliere 1877) as given in *La France Médicale*, No. 79, 1877.

For many years M. Pierret has studied locomotor ataxy in point of view of localization. He had already reached the result that the posterior column of the cord, in man, must be divided into two regions, physiologically distinct, the median bundle and the radicular zones. To the latter appertain all the tabetic phenomena, properly speaking, the fulgurant pains, the anæsthesia, and the inco-ordination of movement; to the former, a sort of flaccid paralysis of the lower members, rendering the upright position difficult or impossible. Locomotor ataxia is, therefore, a symptom, a systematic affection, in the sense that it attacks certain parts of the cord constituting an anatomical system, that of the sensory fibres. It may arise at any part of this system whatever, it is therefore a good idea to seek to find in the entirety of the nervous system those regions which physiologically represent the posterior radicular zones of the spinal axis. For this purpose M. Pierret has given in this *brochure* an anatomical and clinical study of the trigeminus.

The trigeminus nerve, in its soft or sensory portion, must be considered as representing the posterior roots of nearly all the motor nerves of the face. Indeed, as M. Pierret shows, this sensory root takes its rise on the prolongation of the posterior radicular zones of the spinal cord. On the